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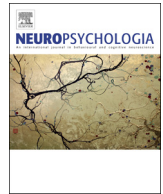
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Neural markers of memory consolidation do not predict temporal estimates of encoded items

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ABSTRACT

In contrast to the paradigms used in most laboratory experiments on interval timing, everyday tasks often involve tracking multiple, concurrent intervals without an explicit starting signal. As these characteristics are problematic for most existing clock-based models of interval timing, here we explore an alternative notion that suggests that time perception and working memory encoding might be closely connected. In this integrative model, the consolidation of a new item in working memory initiates cortical oscillations that also signal the onset of a time interval. The objective of this study was to test whether memory consolidation indeed acts as the starting signal of interval timing. Participants performed an attentional blink task in which they not only reported the targets, but also the estimated target onsets, allowing us to calculate estimated lag. In the attentional blink task, the second target (T2) in a rapid serial visual presentation is often not reported when it follows quickly after the first target (T1). However, if this fast T2 is reported, memory consolidation of T2 is presumably delayed. Consequently, if memory consolidation determines interval onset, we would expect a later estimated onset when consolidation is delayed. Furthermore, as the P3 ERP component is assumed to reflect memory consolidation, we expect that the estimated onsets and subjective lag are functions of the P3 latencies. The behavioral data show that the presumed delay in memory consolidation did not lead to later estimated onsets. In addition, the EEG results suggest that there was no relationship between P3 latency and subjective lag or estimated onset. Overall, our results suggest that there is no direct link between the encoding of items in working memory and sub-second interval timing of these items in the attentional blink task.

1. Introduction

Timing is an indispensable part of our system of cognitive functions. The temporal precision with which sequences of actions are undertaken is crucial in reaching our goals in complex tasks. Often, however, the tasks do not require time perception explicitly. One situation that illustrates the automaticity with which we use timing is contactless payments with bank- or credit cards or electronic devices. We quickly learn how long we need to hold our card or devices next to the scanner for a successful transaction. Our experience with this method of payment teaches us how long this interval is supposed to last and, importantly, makes us able to set off alarm bells when the transaction is taking too long, indicating that something is wrong with either our payment or the device.

Humans can distinguish remarkably well between time intervals of different lengths. Several theories have emerged that model how we keep track of time in the seconds to minutes range. One of the most influential cognitive models of interval timing is the Scalar Timing

Theory (STT; Gibbon, 1977; Gibbon et al., 1984). STT proposes that timing behavior in humans and animals is controlled by three processes: the clock, memory, and decision process. The clock process consists of a pacemaker that outputs pulses at a regular rate and an accumulator that assembles these pulses over time. In between, there is a gate, or switch, component that allows the pulses to reach the accumulator when a salient starting signal is provided. The accumulated amount of pulses can be stored in memory and decisions about durations can be made by comparing the output of the accumulator with other interval durations stored in memory. Although the STT model does not make any assumptions about its neurobiological implementation, it has proven difficult to map its processes to specific brain mechanisms (van Rijn et al., 2014).

Whereas STT focuses on a description of interval timing at a functional level, the striatal beat-frequency (SBF) model offers a model of the internal clock that is inspired by neurobiological mechanisms (Matell and Meck, 2004). In this way, an extended version of the SBF model (van Rijn et al., 2014) can explain the most important behavioral

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findings, while also making explicit assumptions about the neural mechanisms that implement the timing behavior and, roughly, in what brain regions these functions are carried out. In the SBF model, groups of neurons in the cortex act as oscillators that have their own stable oscillating frequency. Through the influence of dopaminergic input, salient events can act as the starting signal by resetting the phases of these oscillators. Because the oscillators have their own frequency, the oscillators will drift out of phase after onset and this will result in predictable patterns of desynchronization corresponding to the time that has passed. The SBF model proposes that these patterns are then detected by medium spiny neurons in the striatum (Buhusi and Meck, 2005). In this way, for example, a specific group of striatal neurons always becomes active 2 s after the phase reset, while another group activates generally after 3 s.

Independent of the actual implementation, however, these models of interval timing face two problems in explaining timing in the real world, in which most forms of interval timing happen continuously and implicitly (Taatgen et al., 2007). First, they can only time a single interval and predict that timing multiple events is difficult (van Rijn and Taatgen, 2008), whereas complex real-world tasks require keeping track of several simultaneous intervals. That is, humans are often faced with keeping track of different concurrent tasks that have their own implicit temporal regularities. Second, these internal clock models require an explicit starting signal. The onset and offset of an interval need to be consciously perceived in order to observe the passage of time, requiring attention to be directed to the events that mark the interval. For example, during the contactless payment described earlier, the customer is not anticipating a timing task when initiating the payment, as typically the payment will proceed before attention was drawn to the lack of confirmation. Nevertheless, as humans can detect that the transition takes too long, elapsed time must be actively monitored as it progresses and be compared to interval lengths stored in memory to notice that at some point one knows for sure the payment has not succeeded. We therefore have to keep track of how much time has elapsed since the introduction of the card until the point we hear a beep indicating the confirmation, while at the same time, for example, registering the temporal patterns in the social talk that is exchanged with the cashier.

Thus, whereas real-world timing often involves continuous, automatic tracking of multiple intervals, most traditional models of time perception propose a dedicated single clock that requires an explicit starting signal. As a potential solution to this problem, Gu et al. (2015) proposed that interval timing and working memory might be based on the same underlying oscillatory dynamics. It has been suggested that working memory consists of gamma oscillations entrained within theta oscillations (Lisman and Idiart, 1995; Lisman, 2010). In this oscillatory model of working memory, multiple items can be stored in memory through the reactivation of multiple gamma cycles within the slower theta cycles. Gu et al. (2015) proposed that, through multiplexing, this system of updating the semantic information of working memory can also code for time. In this way, each item in working memory has an associated oscillatory state that can be detected by medium spiny neurons, as proposed by the SBF. In contrast to STT-like models, including SBF, this integrative model does not require an explicit starting signal. Instead, the consolidation of a memory trace in and of itself makes it possible to estimate the time that has passed since an event was encountered. In addition, the nested oscillations in the working memory model allow temporal information of multiple items to stay active concurrently.

The integrative theory of time perception predicts that there is a tight link between the consolidation and maintenance of items in working memory and time perception. Indeed, there is cumulative evidence that working memory representations and working memory load influence subjective time perception (see Matthews and Meck, 2016 and Gu et al., 2015 for extensive reviews of the relation between memory and timing). For example, visual stimuli that match the

features of active working memory representations are judged to be longer (Pan and Luo, 2012). One specific prediction of the integrative theory of time perception, however, has as of yet remained unexplored: temporal estimations should be based on the moment when an item is encoded in working memory. Here, we will test this hypothesis using the attentional blink (AB) paradigm, in which the temporal aspects of memory consolidation are well recorded and traceable through electrophysiological markers.

Participants in an AB study are shown a rapid serial visual presentation (RSVP), a fast stream of stimuli in the same location on a screen. Within this stream of stimuli, one or two targets are embedded. The task of the participant is to remember these targets and report them after each trial. The well-documented AB phenomenon arises when the second target (T2) is not correctly reported when it is presented in a period of ~ 200–500 ms after the first target (T1) (Raymond et al., 1992; Vogel and Luck, 2002). This effect has been ascribed to a two-stage process, in which after an initial detection and identification stage, targets have to be consolidated in a memory system with limited, serial encoding capacity in order to create a stable representation that is available for report (e.g. Chun and Potter, 1995; Jolicoeur and Dell'Acqua, 1998; Akyürek et al., 2017). Therefore, while T1 is consolidated, a subsequent target (T2) cannot be processed in working memory, thereby causing an AB.

In order to study the processing of targets in the AB task, the P3 component of the averaged event related potential (ERP) has been used as a measure of the latency of memory consolidation (Vogel and Luck, 2002; Kranczioch et al., 2003). The P3 is sometimes referred to as the P300, because its onset generally occurs ~300 ms after the presentation of a salient target, although the range depends on factors such as modality and task conditions (Polich, 2007). It has been shown that targets presented within the window of the AB elicited a P3 when they were reported, but not when the targets were blinked (Vogel et al., 1998). In contrast, other ERPs related to early visual processing are still present when the second target is blinked. Therefore, the P3 component has been associated with memory consolidation (Donchin, 1981; Vogel et al., 1998; Kok, 2001; Akyürek et al., 2010).

Interestingly, second targets that are correctly identified despite being presented within the AB window do elicit a P3, but this P3 exhibits an increased latency and variability. For example, Vogel and Luck (2002) reported the P3 of T2 during the AB window to be ~100 ms later than the P3 of a T2 that was presented outside the AB window, suggesting that this reflects delayed memory consolidation (see also Sessa et al., 2007; Martens et al., 2006). In addition, using single-trial ERP analyses, Chennu et al. (2009) reported that the P3 associated with T2 presented in the AB window were not only delayed, but also showed more temporal variation compared to trials with a longer T2 lag. Thus, these studies show that the timing of working memory consolidation is delayed and more variable when the second target follows quickly after the first.

In the current study, we will utilize these phenomena associated with T2 encoding to test whether memory consolidation determines the onset of temporal estimation. Specifically, we will investigate if the perceived time interval between T1 and T2 in an AB task will indeed be longer if the P3 component for T2 is delayed. Two AB experiments were conducted to explore this question. Crucially, in both experiments, participants not only report target identity, but also the perceived temporal positions of the targets. In Experiment 1, we will test the relation between encoding and time perception behaviorally, by comparing the perceived duration between targets in trials in which T2 is presented within the AB window with trials in which T2 is presented outside this window. We expect that the temporal estimations of the second targets that are presented in the AB window will be delayed compared to the estimations for targets outside of this window. As a result, we also expect that the estimated lag between T1 and T2 will be larger within the AB window. By measuring EEG in Experiment 2, we will compare the latency of the P3, as an electrophysiological index of

memory consolidation, with temporal estimations. We hypothesize that an increased P3 latency associated with a target will result in a later temporal estimate for that target.

2. Experiment 1

2.1. Methods

2.1.1. Subjects

Forty-five participants were recruited for the first experiment (25 female, mean age: 20.7, SD: 2.1). One participant was excluded from the analysis due to a technical malfunction. All participants were recruited from the University of Groningen Psychology Participant Pool and received partial course credits for participating. The study was approved by the Psychology Ethical Committee of the University of Groningen (15163-NE), and participants gave informed consent before testing.

2.1.2. Stimuli and apparatus

Each trial consisted of an RSVP followed by a response screen, in which participants indicated the identity and temporal location of the targets in the RSVP. The stimuli and response screen were presented on a 19 in. CRT monitor with a resolution of 800×600 px and a refresh rate of 100 Hz, using Psychophysics toolbox running on Matlab 2015b under Windows 7. The alphanumeric characters in the RSVP were presented in Courier font in white on a black background with an average size of 0.8° visual angle. Subjects estimated the temporal position of the first target by clicking on a line presented on the left half of the screen and of the second target on a line on the right half of the screen. Both lines were presented simultaneously in white and spanned 320 pixels each (Fig. 1). The horizontal position of the subjects' mouse clicks on the lines were transformed into relative temporal position within the stream of characters. Responses to the identity of the targets were recorded with a computer keyboard.

2.1.3. Procedure

Participants were instructed that they would see trials consisting of one or two letters within a fast stream of numbers and that their task was to report which letters they had seen and when they had seen them. Each trial consisted of an RSVP preceded by a 1000 ms fixation cross, and followed by a response screen (Fig. 1). The RSVP consisted of 21 characters, presented 100 ms each. T1 and T2 were drawn from capital letters from the alphabet (A, B, C, D, E, F, H, J, K, P, R, T, U, V), the distractors were drawn from the digits 1–9.

A total of 300 trials was presented, divided into 6 equal size blocks. In addition, 10 practice trials were presented at the start of the

experiment. In 3% of the trials no target was presented, in 16% of the trials only one target was presented and in 81% of the trials two targets were presented. T1 was always presented as the 4th, 5th, 6th, 7th or 8th character in the stream. T2 was presented lagging 1, 3, 5, 7 or 9 positions behind T1. Each combination of T1 position and T2 lag was presented equally often.

After the RSVP, a response screen was shown. First, participants indicated the temporal positions of the targets relative to the entire stream. They did so by clicking on a line which length represented the duration of the RSVP. Second, responses were made pertaining to the identity of T1 and T2. Subjects were instructed to click the "Second target not seen" button if they had not seen T2. The experiment script is available at: <http://osf.io/54xuj/>.

2.1.4. Analysis

To analyze T2 accuracy and the temporal estimations, we fitted linear mixed models (LMMs) using the *lme4* package in R (Bates et al., 2014). We performed model comparisons using likelihood ratio tests to evaluate whether a fixed factor improved the model fit. In these tests, subject was always included as a random intercept term. Only fixed factors that improved the model fit were included in the final model. Next, we gradually increased the random effect structure of this model by adding random slopes for the significant fixed factors and comparing the more complex model with the simpler model using a likelihood ratio test. In the case of multiple potential random slopes, we first added the random slope that improved the model fit most, at every step. To this end, we determined which random slope led to the model with the lowest AIC. A random slope term was only included if it improved the model significantly and the statistics of the fixed factors of the best model are reported here.

To quantify the evidence in favor of the null hypothesis compared to the alternative hypothesis we calculated Bayes factors. For the binomial models fitting the accuracy data, we approximated the Bayes factor using the BIC values of H_0 and H_1 , as described in Wagenmakers (2007). For the temporal estimation models, Bayes factors were calculated using the *lmBF* function from the *BayesFactor* package in R (Morey et al., 2014). We will denote the evidence for the null hypothesis (H_0) over the alternative hypothesis (H_1) as BF_{01} .

2.2. Results

2.2.1. Attentional blink

The mean accuracy for T1 and for T2 given correct report of T1 is shown in Fig. 2A. A binomial linear mixed model was estimated with accuracy of T2 as the dependent variable, lag as a categorical fixed factor and subject as a random factor. The inclusion of lag improved the

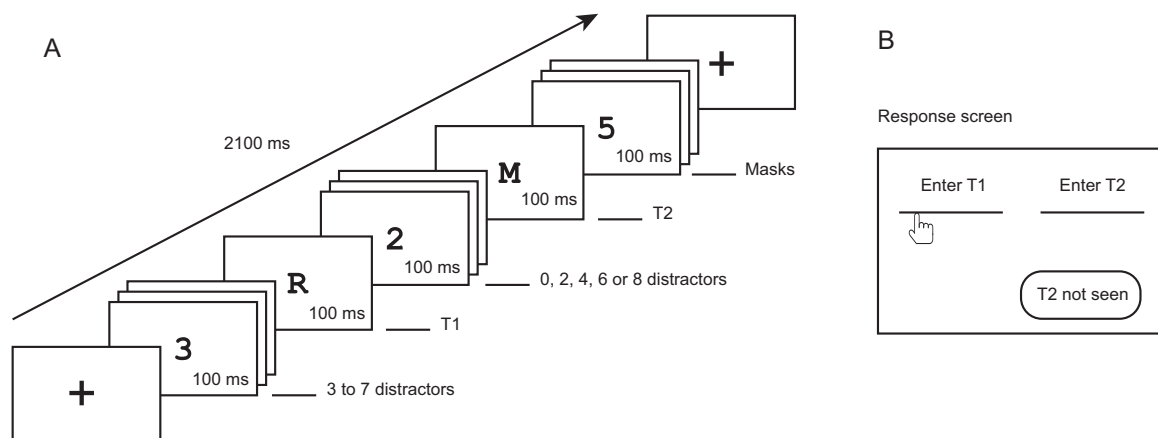


Fig. 1. Overview of a trial in Experiment 1. A trial consisted of an RSVP with 0, 1 or 2 target letters in a stream of distractor digits (Figure A). A fixation cross was presented before and after the trial. At the end of the trial, participants saw a response screen in which they reported the estimated onset of the two targets on a line representing the trial duration (Figure B). In addition, they reported the target identity.

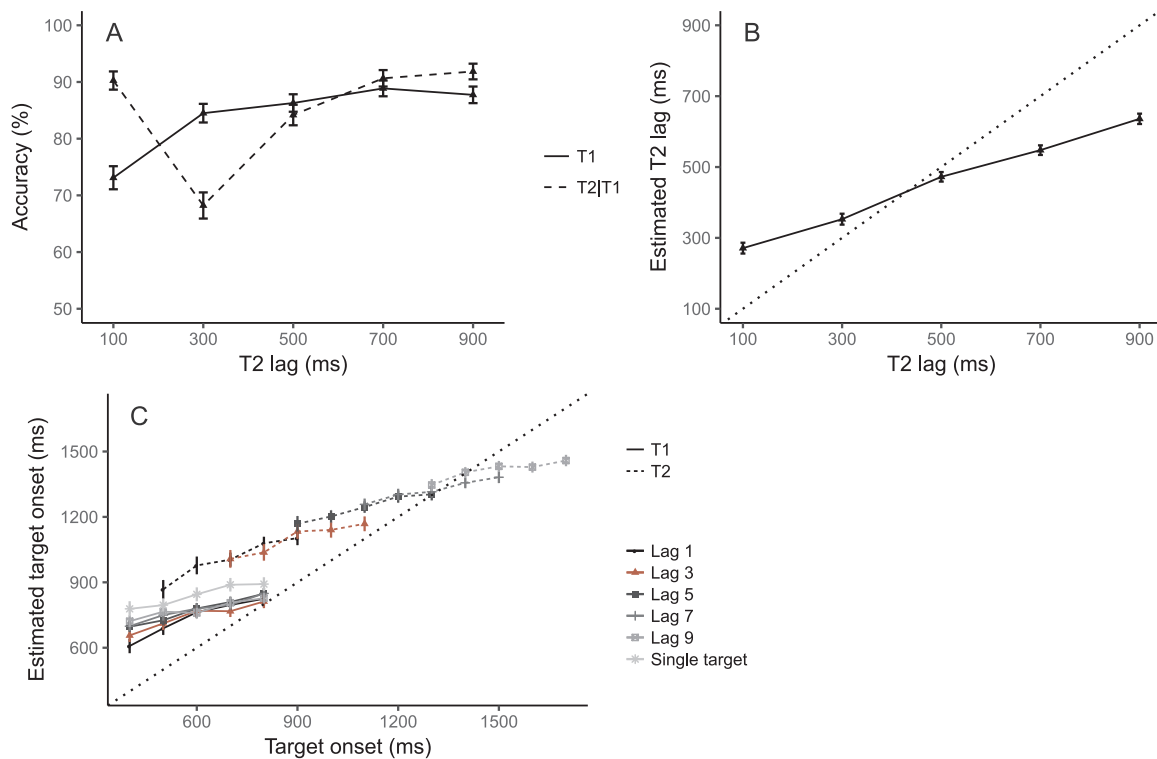


Fig. 2. Behavioral results of Experiment 1. Figure A shows the average accuracy of T1 and T2/T1 per presented T2 lag, demonstrating that there is an attentional blink at lag 3 and lag-1 sparing. Figure B shows the average estimated lag as a function of the presented lag. The dashed line represents veridical estimation. Figure C presents the average estimated target onset as a function of target onset, relative to trial onset. Error bars represent the within-subject CIs.

model significantly ($\chi^2 = 522.40$, $p < 0.001$, $BF_{01} < 0.001$). A post-hoc Tukey's HSD test showed that T2 accuracy was lower at lag 3 than at all other lags ($ps < 0.001$), indicating that an attentional blink occurred (see Table S1 for full Tukey's HSD test results). Also, T2 performance at lag 1 was significantly better than at lag 3 ($p < 0.001$), indicating lag-1 sparing (Potter et al., 1998).

2.2.2. Temporal estimations

We calculated the estimated T2 lag as the difference between the estimated T1 position and the estimated T2 position. The average estimated T2 lag for each presented T2 lag is displayed in Fig. 2B. A linear mixed model was fitted with estimated lag as the dependent variable and the centered presented lag as a continuous fixed factor. Subject was included as a random intercept term and lag as a random slope term. The model revealed an unstandardized coefficient of presented lag of $\beta = 0.45$ ($t = 13.80$, $p < 0.001$, $BF_{01} < 0.001$), showing that lag estimation increased linearly with presented lag. As can be seen in Fig. 2B, responses to relatively short intervals tended to be overestimated and responses to relatively long intervals tended to be underestimated. To test if there was a significant pull towards the (subjective) mean, a linear mixed model was estimated with estimation bias (the difference between the presented lag and the estimated lag) as the dependent variable, presented lag as a continuous fixed factor, subject as a random intercept term and lag as a random slope term. Whereas a coefficient of 0 would indicate perfect estimation, the model yielded a coefficient of $\beta = -0.55$ ($t = -16.88$, $p < 0.001$, $BF_{01} < 0.001$). Post-hoc, we tested the possibility that the response format could lead to compressed lag estimations when T1 occurred later in the stream. To this end, we added T1 position to the model predicting estimated lag, but found that this did not improve the model fit ($\chi^2 < 0.01$, $p = 0.973$, $BF_{01} = 40.994$). This finding indicates that lag estimations did not depend on when the targets appeared in the stream.

Thus, estimated lag increased linearly with presented lag, but the estimations showed a significant pull towards the (subjective) mean. To

test if lag estimations increased when T2 was presented within the window of the AB, we added the dichotomous factor "within AB window" to the model predicting estimated lag. The results showed that lag estimates for lag 3 were not longer than lag estimates outside the AB window. Instead, a trend in the opposite direction was observed ($\beta = -0.167$, $t = -1.94$, $p = 0.052$, $BF_{01} = 6.612$).

In addition, we hypothesized that when T2 was presented within the window of the AB (i.e., lag 3), the delay in memory consolidation would lead to later temporal estimations of T2. Fig. 2C shows the temporal estimates of T2 for each T2 position and lag. If temporal estimates would be delayed in lag 3, we expected a relatively high intercept for the line corresponding to lag 3 compared to the other lags in this figure. To test whether T2 estimates were indeed delayed at lag 3, an LMM was estimated with temporal estimation of T2 as the dependent variable, actual temporal position of T2 as a continuous fixed factor, subject as a random factor and the random slope of temporal position of T2. Adding lag to the model as a categorical fixed factor yielded a significant improvement ($\chi^2 = 61.47$, $p < 0.001$, $BF_{01} < 0.001$). However, the model showed that T2 estimates at lag 3 were *earlier* than at lag 5 ($\beta = -0.75$, $t = -7.15$, $p < 0.001$), lag 7 ($\beta = -0.80$, $t = -6.30$, $p < 0.001$), and lag 9 ($\beta = -0.96$, $t = -6.14$, $p < 0.001$).

Fig. 2C also shows the temporal estimates for T1 for each T1 position and lag. An LMM was estimated with temporal estimation of T1 as the dependent variable, actual temporal position as a continuous fixed factor and lag as a categorical fixed factor, subject as a random intercept and lag as a random slope term. Again, a pull towards the mean was demonstrated by the unstandardized estimate of the fixed factor ($\beta = 0.37$, $t = 22.02$, $p < 0.001$, $BF_{01} < 0.001$). Including lag in the model increased its descriptive value ($\chi^2 = 133.50$, $p < 0.001$, $BF_{01} < 0.001$), showing that the temporal estimation of T1 was also affected by if and when T2 was presented. A post-hoc Tukey's HSD comparison of the T2 lag conditions revealed that T1 estimations in single target trials were later than in two target trials ($ps < 0.001$) (see Table S2 for an overview of the test results). To further investigate the

effect of T2 on T1 estimations, we post-hoc estimated an LMM predicting T1 estimation in single target and lag 3 trials (see also Fig. S1). We found that T1 estimations were earlier when a second target was reported compared to when only one target was reported ($\beta = -0.86$, $t = -5.70$, $p < 0.001$, $BF_{01} < 0.001$), regardless of whether this T2 was reported correctly or not ($\chi^2 = 0.06$, $p = 0.799$, $BF_{01} = 2.973$) or whether it was a single target trial or a lag 3 trial ($\chi^2 = 2.43$, $p = 0.119$, $BF_{01} = 6.450$). These findings indicate that merely reporting two targets led to earlier temporal T1 estimates. All analysis scripts and data are available at: <http://osf.io/54xuj/>.

2.3. Discussion

In Experiment 1, we aimed to test whether time estimations of T2 are delayed if memory consolidation is delayed. We found a lower accuracy for T2 at lag 3 compared to longer lags, indicating that an AB was present. In line with previous studies, we therefore assume that a delay was induced in the latency of memory consolidation of T2 in the lag 3 condition, compared to lags outside the AB window (Vogel and Luck, 2002; Chennu et al., 2009).

We demonstrated that temporal estimations of T2 lag increased with presented T2 lag, but also showed a linear pattern of underestimations of the longer lags and overestimation of the shorter lags. This pull towards the mean effect is typically observed in experiments in which intervals have to be reproduced in a specific temporal context (Grondin, 2001; Jazayeri and Shadlen, 2010). In contrast to our hypothesis, temporal estimations of lag and T2 in the lag 3 trials were not later than temporal estimations in the other lag conditions: T2 was estimated to have occurred relatively early when it was presented within the AB window compared to later windows. Thus, whereas memory consolidation might have been delayed in lag 3 trials, this did not lead to a delay in temporal estimation.

In summary, the results do not support the hypothesized role of memory consolidation as the index of timing. Importantly, however, the current behavioral experiment can only give us indirect evidence about the link between memory consolidation and temporal estimation. Although we assume that memory consolidation of T2 is delayed in lag 3 trials, this assumption cannot be verified on the basis of purely behavioral data. In addition, the current experimental setup does not allow for taking inter-trial variation in memory consolidation - which could explain inter-trial variation in temporal estimates - into account. In Experiment 2, we will address these issues by measuring EEG during the AB task.

3. Experiment 2

An EEG study was conducted to further investigate if the latency of memory consolidation influences time perception. It has been suggested that the P3 component of the ERP is a neural correlate of memory consolidation (Donchin, 1981; Vogel et al., 1998; Kok, 2001). In Experiment 2, we will therefore use P3 latency as an index of delay in memory consolidation.

We investigated the relationship between P3 latency and temporal estimations in two ways. First, we compared the P3 latencies of relatively "early" and "late" estimations by creating a median split based on the temporal estimations of correctly identified T2s in the lag 3 condition. Second, using single-trial ERP analysis, we assessed whether there is a relation between inter-trial variation in P3 latency and temporal estimations. We expected that 1) the P3 for T2 will exhibit an increased delay and variability in lag 3 trials compared to trials in which the lag is longer, and 2) inter-trial variation latency in the P3 can account for variation in the temporal estimations. Specifically, we expected that in trials where a T2-elicited P3 was relatively early, the subjective estimation of T2 was also relatively early.

3.1. Methods

3.1.1. Subjects

Thirty subjects participated in Experiment 2 (21 female, mean age: 22.2, SD: 2.4). Nine participants were recruited from the University of Groningen Psychology Participant Pool and received partial course credits for participating, 21 participants were recruited via social media and were rewarded 15 euro. The study was approved by the Psychology Ethical Committee of the University of Groningen (15163-NE), and participants gave written informed consent before testing.

3.1.2. Stimuli and apparatus

The same setup of stimuli presentation was used as in Experiment 1.

3.1.3. Procedure

A procedure similar to Experiment 1 was employed. However, following Chennu et al. (2009), the rapid serial stream of stimuli consisted of 35 characters, presented 100 ms each. In total, 312 trials were presented, divided into 6 equal size blocks. T1 was preceded by 4–16 characters in the stream. T2 was presented in 75% of the trials, lagging 1, 3 or 8 positions behind T1. Each combination of T1 position and T2 lag was presented equally often. Additionally, participants received feedback on the accuracy of target identity and their temporal estimations in the 10 practice trials preceding the experiment. The experiment script is available at: <http://osf.io/54xuj/>.

3.1.4. EEG recording

EEG was recorded using a WaveGuard EEG cap electrode cap (eemagine Medical Imaging Solutions GmbH, Berlin, Germany) and a TMSi amplifier (Oldenzaal, The Netherlands). Impedance was reduced to less than 15 K Ω . Electrical signals were measured at 1000 Hz from 23 electrodes placed at the following locations in the international 10/20 system: Fp1, Fp2, Fz, F3, F4, F7, F8, FCz, FC1, FC2, Cz, C3, C4, T7, T8, Pz, P3, P4, P7, P8, Oz, O1, O2. Horizontal eye movement was measured with two electrodes placed by the participants' canthi, vertical movement was measured with two electrodes above and below the right eye.

3.1.5. EEG pre-processing

Raw EEG data was preprocessed and analysed with EEGLAB 13.5.4b, ERPLAB 5.0.0.0, custom Matlab scripts and R. A bandpass filter was applied to the raw data with a 25 Hz high cut-off and 0.1 Hz low cut-off frequency. The EEG data was re-referenced to the average of the left and right mastoids. Independent component analysis was performed on each recording. After inspection of scalp topography, components associated with ocular movement were removed and remaining components were back-projected (Hoffmann and Falkenstein, 2008). Epochs were created for each presented T1 starting 500 ms before the target was presented and ending 2500 ms after the target appeared. Baseline correction was applied using the 200 ms period before the target as the baseline period. Automatic artifact rejection was applied to epochs containing samples that exceeded the threshold of 150 μ V. Following, for example, Chennu et al. (2009), Sessa et al. (2007) and Martens et al. (2006), all analyses were based on the EEG signal at the Pz electrode.

3.1.6. P3 latency estimation

3.1.6.1. Average. A grand average ERP was calculated for trials in which T1 and T2 were reported correctly. To test whether the delay in memory consolidation was different between lags, we estimated P3 latencies per lag for each participant. First, because the P3 is a relatively low-frequency component, we applied a 3.5 Hz low-pass filter to the EEG signal (Jaskowski and Verleger, 2000). Next, P3 latency was estimated as the local maximum within a particular window in the average ERP of each lag condition for each participant. For the P3 associated with T1, this window was 200–600 ms after T1 onset. Visual inspection of the grand average

ERPs per subject showed that the P3s associated with T2 occurred slightly later than for T1. To capture these peaks, for T2 at lag 3 and 8, the time windows were 650–1050 ms and 1150–1550 ms after T1 onset, respectively.

3.1.6.2. Median split. To contrast P3 latencies of targets that were estimated relatively early and targets estimated relatively late, we split the latency estimations of the participants in two groups based on the median estimation. Specifically, the median split was based on temporal estimation bias and was performed for each participant separately on trials where T1 and T2 were correctly identified. First, to correct for the pull towards the mean effect (i.e., the consistent overestimation of the early targets and underestimation of late targets), the temporal estimation bias was corrected for temporal position. Second, we estimated a linear mixed model with temporal estimation of T2 as the dependent variable, actual temporal position of T2 as a fixed factor and subject as a random factor. The amount of deviation of each T2 estimation from the estimated model then gives a corrected value of estimation latency. Third, the median split was performed on the resulting residuals for each participant.

For each participant, lag and target, two average ERPs were calculated, corresponding to trials where the target was estimated to have occurred earlier or later than the median estimation. For peak detection, we used the same time windows and method as in the analysis of the grand averages, described in the previous section.

3.1.6.3. Single-trial. In order to investigate the relationship between memory consolidation and temporal estimations on a single-trial level, we estimated the P3 latency in every trial in which T1 and T2 were correctly identified for every participant. First, we applied a 3.5 Hz low-pass filter to the EEG signal (Jaskowski and Verleger, 2000). Second, for each trial, a linear detrend function was applied. Third, to estimate the latency of the P3, we cross-correlated a peak template with the EEG signal. Following Jaskowski and Verleger (2000), the template consisted of a positive half-cycle sinusoid of 300 ms. Peak latency was determined as the time point at which the correlation was highest within a particular time window. The same windows as in the average peak estimation were employed (see Section 3.1.6.1). To filter out trials in which no peak could be distinguished, only those trials in which the correlation between the template and the signal exceeded 0.65 were included in the analysis. In addition, only trials in which the difference between T1 and T2 peak latencies was greater than 100 ms were considered. In this way, 19% of the trials were excluded, resulting in a total of 1313 single target trials, 1665 lag 8 trials, and 962 lag 3 trials.

All analysis scripts and data are available at: <http://osf.io/54xuj/>.

3.2. Results

3.2.1. Behavioral results

3.2.1.1. Attentional blink. The mean accuracy for T1 and for T2 given correct report of T1 is shown in Fig. 3A. A binomial linear mixed model was estimated with accuracy of T2 as the dependent variable, lag as a categorical fixed factor, subject as a random intercept term and lag as a random slope term. The inclusion of lag yielded a significantly better model ($\chi^2 = 786.80$, $p < 0.001$, $BF_{01} < 0.001$). A post-hoc Tukey's HSD test showed that T2 accuracy was lower at lag 3 than at lag 8, indicating that an attentional blink occurred (see Table S3 for full Tukey's HSD test results). Also, T2 performance at lag 1 was significantly better than at lag 3 ($p < 0.001$), indicating lag-1 sparing.

3.2.1.2. Temporal estimations. Average lag estimations for each presented T2 lag and mean estimation corresponding to each target position are displayed in Fig. 3B and C, respectively. A similar analysis of temporal estimations was conducted as in Experiment 1. A linear mixed model was fitted with estimated lag as the dependent variable and the centered presented lag as a continuous fixed factor. Subject was

added as a random intercept term and presented lag as a random slope term. The model revealed an unstandardized coefficient of $\beta = 0.56$ ($t = 15.08$, $p < 0.001$, $BF_{01} < 0.001$), showing that the lag estimations increased linearly with the presented lag. Adding the dichotomous factor "within AB window" did not improve the model, suggesting that temporal estimates were not delayed for lag 3 compared to lag 1 and lag 8 ($\chi^2 = 0.38$, $p = 0.535$, $BF_{01} = 35.536$). Similar to Experiment 1, a linear mixed model was estimated with bias in lag estimation as the dependent variable, presented lag as a continuous fixed factor and subject as a random intercept factor and lag as a random slope term. This model showed that there was a significant pull towards the mean ($\beta = -0.44$, $t = -11.63$, $p < 0.001$).

To test whether T2 estimates were delayed at lag 3, an LMM was estimated with temporal estimation of T2 as the dependent variable, temporal position of T2 as a continuous fixed factor, lag as a categorical fixed factor, subject as a random factor and the random slopes of temporal position of T2 and lag. Including lag improved the model fit ($\chi^2 = 24.61$, $p < 0.001$, $BF_{01} = 0.039$). The model showed that T2 estimates at lag 3 were relatively early compared to lag 1 ($\beta = -0.48$, $t = -3.56$, $p < 0.001$), but there was no significant difference between lag 3 and lag 8 ($\beta = 0.29$, $t = 1.94$, $p = 0.052$).

3.2.1.3. Replication experiment. We have replicated the behavioral results of Experiment 2 in an additional experiment. Please find the results of this experiment in the Supplementary Information.

3.2.2. EEG results

3.2.2.1. Average P3 analysis. Fig. 4A shows the grand average ERPs for lag 3 no-blink and blink trials, and lag 8 and single target trials in which both targets were correctly identified. For lag 3 no-blink and lag 8 trials, two large positive peaks can be observed, corresponding to the two presented targets. As expected, the second P3 was absent in lag 3 blink trials, indicating that T2 was not consolidated in working memory.

To evaluate if the P3 was delayed for T2 in lag 3 compared to T1 in lag 3 and T2 in lag 8, we estimated an LMM with P3 latency as the dependent variable, lag (lag 3 and lag 8) and target (T1 and T2) as categorical fixed factors and subject as a random factor. In the model, the contribution of each subject was weighted by the number of trials on which the subject average in each condition was based, using the *lmer* function in the *lme4* package in R.

The LMM showed that there was no difference in T2 P3 latency between lag 3 ($M = 545.73$ ms, $SD = 113.56$) and lag 8 ($M = 544.53$ ms, $SD = 85.84$) ($\beta = 8.03$, $t = 0.46$, $p = 0.646$, $BF_{01} = 3.555$), suggesting that the P3 for T2 was not delayed when this target was presented within compared to outside the AB window. However, within lag 3 trials, we found that the P3 was delayed for T2 compared to T1 ($\beta = -69.50$, $t = -3.71$, $p < 0.001$, $BF_{01} < 0.001$). Thus, these results suggest that the P3 for T2 within the AB window was delayed compared to the P3 for T1, but not compared to the P3 for T2 outside the AB window. Post-hoc, we compared T1 latency for no-blink and blink lag 3 trials by adding a fixed factor to the model coding for whether T2 was correctly reported, but we found no difference ($\beta = -21.50$, $t = -1.02$, $p = 0.311$, $BF_{01} = 3.903$).

3.2.2.2. Median-split P3 analysis. Fig. 4B and Fig. 4C show the average ERPs for estimates that were earlier and later than the median for lag 3 T1 and T2, respectively. Five linear mixed models were estimated to test whether P3 latencies of the early and late estimations were different for T1 and T2 in the lag 3 and lag 8 conditions and T1 in the single-target condition. In each model, P3 latency was the dependent factor, latency category (early or late) was the fixed factor and subject was entered as a random factor. Again, the contribution of each average data point was weighted by the number of trials on which the average was based.

The model for T2 at lag 3 showed that there was no difference in P3

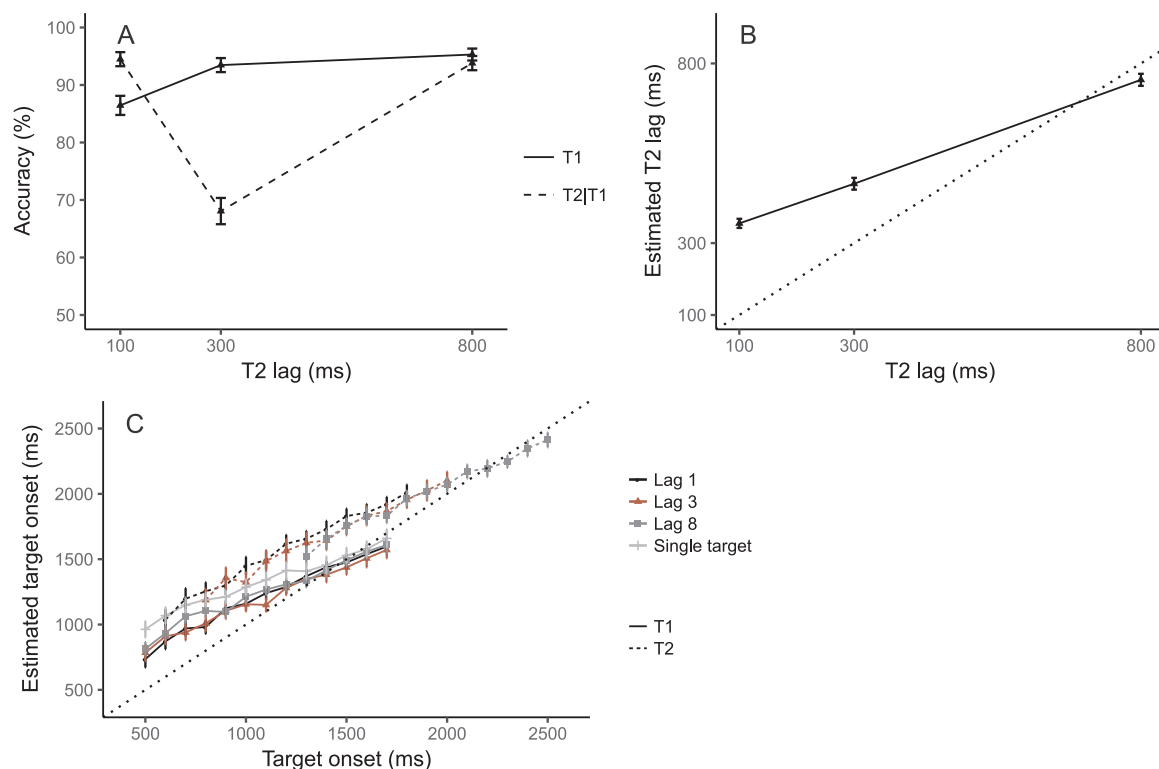


Fig. 3. Behavioral results of Experiment 2. Figure A shows the average accuracy of T1 and T2/T1 per presented T2 lag, demonstrating that there is an attentional blink at lag 3 and lag-1 sparing. Figure B shows the average estimated lag as a function of the presented lag. The dashed line represents veridical estimation. Figure C presents the average estimated target onset as a function of target onset, relative to trial onset. Error bars represent the within-subject CIs.

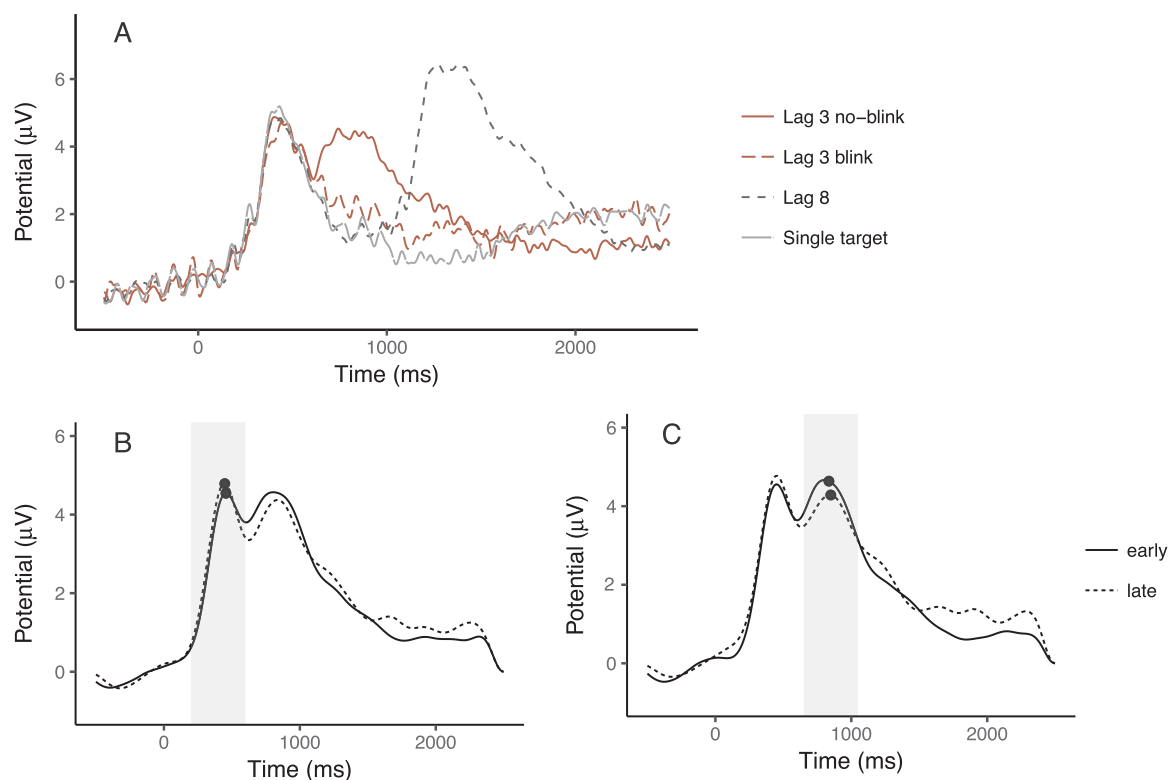


Fig. 4. ERP results of Experiment 2. Figure A shows the grand average ERPs of lag 3, lag 8 and single-target trials in which both T1 and T2 were correctly identified. Figure B and C show the median-split ERPs for early and late estimations of T1 and T2, respectively, in lag 3 trials. The data in Figure B and C has been filtered with a 3.5 Hz low-pass filter. The grey areas show the windows employed for local peak detection. The black dots represent the average local peak for early and late estimations.

latency between early ($M = 835.43$) and late ($M = 849.20$) estimations ($\beta = 10.69$, $t = 0.44$, $p = 0.667$, $BF_{01} = 3.296$). In line with this, we did not find differences in the P3 latencies associated with early and late estimations for T1 for lag 3, lag 8 and single-target trials ($p > 0.596$, $BF_{01} > 2.849$) and for T2 for lag 8 ($\beta = -9.01$, $t = -0.76$, $p = 0.451$, $BF_{01} = 2.24$). Thus, overall, these results suggest that the estimated target onsets did not depend on the latency of the P3. See Table S4 for an overview of the mixed model and Bayes factor results.

3.2.2.3. Single-trial P3 analysis. In our single-trial analysis we first tested whether there were latency differences of P3 associated with T1 and T2 in the different lags. To this end, we estimated an LMM with P3 latency as the dependent variable, lag and target as categorical fixed factors, subject as a random intercept factor and lag and target as random slope terms. The model showed that the P3 associated with T2 occurred later for lag 3 ($M = 570.53$) than for lag 8 ($M = 541.76$) ($\beta = 31.44$, $t = 6.41$, $p < 0.001$). In addition, the P3 was estimated earlier for T1 ($M = 411.38$ and $M = 427.96$ for lag 3 and lag 8, respectively) than for T2 in lag 3 ($\beta = -162.19$, $t = -23.72$, $p < 0.001$) and lag 8 trials ($\beta = -115.82$, $t = -18.92$, $p < 0.001$). Although the standard deviation of T2 P3 latency was only slightly larger for lag 3 ($SD = 109.50$) than for lag 8 ($SD = 107.21$), the standard deviation for T2 P3 in lag 3 was notably larger than for T1 ($SD = 94.78$). Thus, in contrast to average P3 analysis, the single-trial analysis suggests that the T2 P3 was delayed for lag 3 trials compared to lag 8 trials.

Fig. 5A shows the color map for lag 3, which represents the single-trial ERP amplitude ordered by T2 P3 latency. Correspondingly, Fig. 5B shows single-trial temporal estimations (relative to the median for each subject) in the same trial order as Fig. 5A. The regression line in Fig. 5B suggests that there was no clear correlation between T2 P3 latency and the time estimates. To investigate the influence of single-trial P3 latency on time estimations, eight linear mixed models were computed, predicting estimated T1 position in single target, lag 3 and lag 8 trials and estimated T2 position and lag in lag 3 and lag 8 trials. In all models, estimated T1 position was entered as the dependent variable, T1 position and P3 latency were continuous fixed factors and subject was a random factor. For all models, except the lag 3 model with P3 lag as a fixed factor, the random slope of T1 position was included because it improved the model significantly.

In line with Fig. 5, we found no effect of P3 latency on the estimation of T2 ($\beta = 0.03$, $t = 0.28$, $p = 0.777$) and lag ($\beta = -0.03$, $t = -0.49$, $p = 0.625$) in lag 3 trials (see Table S5 for an overview of the mixed model results). The corresponding Bayes factors of 7.349 and 8.961, respectively, suggest that there was moderate evidence (Jeffreys, 1961) for the null hypothesis, i.e. the absence of an effect of P3 latency

on temporal estimates. Over all lags and targets, we did not find a significant positive effect of P3 latency on temporal estimation (Table S5). We did find a small *negative* effect of P3 latency on T1 and lag estimations in lag 8 trials. However, the corresponding Bayes factors ($BF_{01} = 14.76$ and $BF_{01} = 1.26$, respectively) show that there was more evidence for the absence of an effect of P3 latency than for the alternative hypothesis. Thus, overall, the single-trial analysis does not deliver evidence in support of a relationship between P3 latency and temporal estimation.

3.3. Discussion

The goal of Experiment 2 was to relate electrophysiological signatures of memory consolidation to temporal estimations. We expected temporal estimations of identified T2s that were presented within the AB window to have increased latency and variance. If memory consolidation acts as an index of timing onset, we expected that the observed variance in temporal estimations could be explained by the variance of P3 latency.

We found similar behavioral results as in Experiment 1, despite the different combination of temporal positions and lags. In line with Experiment 1, temporal estimations for T2 in lag 3 trials, in which there is a presumed delay in memory consolidation, were not delayed compared to temporal estimations outside the AB window. Whereas the average ERP analysis revealed only a small, non-significant latency difference between the P3 evoked by T2 inside and outside the AB window, the single-trial analysis showed a ~ 30 ms delay in the lag 3 condition compared to the lag 8 condition. The latter finding is in line with previous studies (Sessa et al., 2007; Martens et al., 2006; Vogel and Luck, 2002; Chennu et al., 2009).

We expected that the latency of memory consolidation, as reflected by the P3, would determine the subjective onset timing of the participants. However, the analysis of the median-split ERPs revealed that there were no differences in P3 latency between early and late estimations. Similarly, we found no positive relation between P3 latency and temporal estimations on a single-trial level.

4. General discussion

This study was aimed at finding evidence of the involvement of memory consolidation in the starting of the internal clock in interval timing. We made use of the AB task, as it has been shown that memory consolidation is delayed and more variable when a target is presented within the AB window (Sessa et al., 2007; Martens et al., 2006; Vogel and Luck, 2002). If memory consolidation serves as the onset of interval

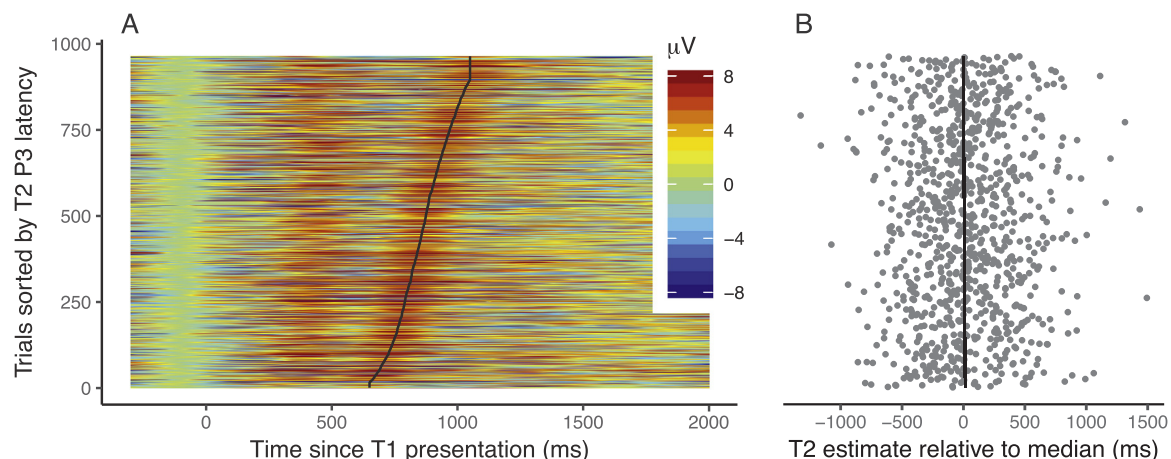


Fig. 5. Single-trial P3 results of Experiment 2. Figure A shows the color map for lag 3 trials in which both T1 and T2 were identified correctly. The ERP amplitude of the trials are displayed sorted by T2 P3 latency. The black line represents the estimated single-trial P3 latency. Figure B shows the estimated temporal estimations of the target relative to the subject median for each trial. The trials are displayed in the same order as in Figure A.

timing, we expected that temporal estimations would be delayed when memory consolidation is delayed. In addition, we expected that inter-trial variation in temporal estimations could be explained by variation in memory consolidation.

In Experiment 1, we showed that participants could estimate the timing of the two targets in a predictable way, in which the temporal estimates increased with the presented onset. However, in line with typical temporal reproduction tasks, short lags were overestimated and long lags were underestimated (Grondin, 2001; Jazayeri and Shadlen, 2010; van Rijn, 2016). Although a classic AB was observed, temporal estimations of identified T2s were not affected by the hypothesized delay of memory consolidation. Instead of temporal estimations of T2 being later when memory consolidation was delayed, we found that temporal estimations were slightly earlier. In Experiment 2, we found no latency differences between P3s for early and late temporal estimates and no relation between inter-trial variation in temporal estimates and single-trial P3s.

Thus, overall, we found no evidence for a relationship between memory consolidation as indexed by a P3 in an attentional blink task, and the associated temporal estimations. Whereas models of interval timing, such as the Scalar Timing model (Gibbon, 1977; Gibbon et al., 1984; Wearden, 1991) and the SBF model (Matell and Meck, 2004), propose a clock mechanism that an explicit starting signal starts a timing mechanism, Gu et al. (2015) proposed that, through multiplexing, the system of updating the semantic information of working memory can also code for time. In the latter case, working memory consolidation could serve as the "starting gun" that resets the phase of the oscillation coding for a specific item. However, the current study suggests that memory consolidation, as reflected by the P3, might not fulfill this role. It seems that, although memory consolidation is crucial for the conscious perception of an event, time information is coded for by another mechanism that is independent of the processes underlying the P3. However, a recent study suggests that the P3 might play a different role in timing, reflecting norepinephrine release in the overestimation of unexpected stimuli (Ernst et al., 2017).

It is important to note that the current study relies on three assumptions. First, working memory consolidation of T2 is delayed for no-blink trials in which T2 quickly follows T1, and second, the P3 component reflects working memory consolidation in the AB task. We believe that these assumptions are well supported by an extensive body of behavioral and EEG studies (for a review, see Martens and Wyble, 2010). In addition, most theoretical models of the AB assume that the second target is perceived, but fails in a limited-capacity memory system, and the latter process would therefore be delayed when T2 is reported in no-blink trials. Third, we assume the memory consolidation process has a dichotomous outcome: it is either successful or not. Consolidation was considered successful when an item was correctly recalled. However, we make no assumptions regarding the strength of memory consolidation, as was proposed in, for example, Wixted and Mickes (2010). It could be argued that stronger memory consolidation affects interval timing differently than weaker memory consolidation. For example, items that subjects judge to remember may have been consolidated into memory more strongly than the items of which subjects only have a vague idea. In our task, however, the weakly remembered items will often not be recalled correctly, since guessing correctly has only a 1/26 probability (or 1/14 if the participant was aware of the particular letter set used in our experiments).

A potential alternative to our working memory consolidation hypothesis is that timing onset might be established earlier than conscious stimulus detection. In line with this notion, Amano et al. (2016) showed that the threshold of the MEG response related to the point of subjective simultaneity was earlier than the threshold for reaction time. They argued that although stimulus onset is determined prior to stimulus detection, the established time marker is only available when the stimulus is consciously perceived. Indeed, future studies might investigate the possibility that earlier perceptual processes, and associated earlier

perceptual EEG components (e.g. N1 and P2), determine perceived timing.

Although the results suggest that temporal estimates in the sub-second range do not depend on the latency of working memory consolidation, potential shortcomings of the current study have to be considered. First, the variation in P3 latencies found in the AB task might be too small to reliably account for the variation in temporal estimations. Second, the assessment of temporal estimations by clicking on a timeline might not be precise enough to reveal small nuances in perceived timing. Even though the time estimation results show that, on average, the estimates increase linearly with the presented timing, the inter-trial response variation might lead to a decreased accuracy in mapping perceived timing to estimations. In addition, the observed pull towards the mean effect decreases meaningful inter-trial variation related to the presented lag. The response format could also compress the estimates for T2 when T1 appears later in the stream. However, in our models predicting estimated T2 position, any linear effects of position have been accounted for by the inclusion of actual temporal position of T2 as a fixed factor. In addition, our post-hoc test in Experiment 1 showed that lag estimations did not depend on when the targets appeared in the stream. The estimations of T1 and T2 do seem to interact in one particular way, however: we found earlier T1 estimates for trials in which two targets were reported compared to trials in which only a single target was reported.

In summary, the current study suggests that the timing of interval onset is not determined by memory consolidation as operationalized in this study: We found no relationship between neural markers of memory consolidation latency and reported target onset estimations.

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Declaration of interests

None.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.neuropsychologia.2018.04.039>.

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